### C. M. A. SECTION ON PUBLIC HEALTH

Whatever have been the factors and forces which have, until this time, prevented the establishment of a section on Public Health in the California Medical Association, it is now a fact accomplished—its future rests squarely on our shoulders. It may take some time before Health Officers will develop the habit of attending the annual meeting of the Association.

At present, and for many years, the Health Officers' Association of our State has met with the public health department of the League of California Cities. This coöperation will continue because of the close relationship with the governing bodies of the cities of the State.

### HEALTH OFFICERS AND ORGANIZED MEDICINE

A fairly large number of health officers are not members of the California Medical Association, and for this again there are reasons. In some instances, health officers have not felt welcome at the State Association meetings, and in others the health officer has held himself aloof from the Association. In most cases, however, the successful health officer and his associates are active members and integral parts of the Association. If this public health section is to be a success, this relationship must be fostered and developed both by the California Medical Association and the Public Health associations. Every health officer should be an active member of his respective county medical association.

The public health section can do a great deal to enhance the mutual value of the private practitioner of medicine and the public health practitioner. This is emphasized when one stops to realize that, while the department of public health has the immediate responsibility for the control of disease, the most important force in this control is the private practitioner of medicine.

### IN CONCLUSION

As chairman, at this first meeting of the newly-established Section on Public Health, I know I may express the appreciation of our group for the action that led the C.M.A. chairman of the Committee on Scientific Work, Dr. George H. Kress, to secure the approval and consent of the Council of the California Medical Association to establish this additional scientific section. The excellent attendance at this initial session is most gratifying and bespeaks increasing interest for the sessions to be held in years still ahead.

703 California State Building.

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*Rudolf Virchow (1821-1902).*—In the history of pathology there is no more dominant figure than that of Rudolf Virchow. His book, "Cellular-Pathologie," established the doctrine of cellular pathology, and marked a new high in the advance of modern scientific medicine. Military sanitation was another field in which Virchow did outstanding work. However, although he was intensely interested in the medical aspects of war, he deplored the fact that wars should be necessary. His writings on the subject show that he labored valiantly in the cause of lasting and universal peace.—Warner's *Calendar of Medical History.*

## LEUKEMIA AS A CAUSE OF NASAL OBSTRUCTION

### REPORT OF CASE

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CASES of leukemia in which infiltration or tumor masses have localized in the nasal passages and progressed to cause obstructive symptoms are apparently very rare. In 1925, Seelenfreund[1] reviewed the literature of leukemic changes in all parts of the upper respiratory tract and found that forty cases has been reported since 1845. Those few reports that concerned changes in the nasal mucosa described a pale lesion with intact epithelium beneath which a leukemic stroma infiltrated about and into blood vessels and ducts, replacing the normal connective tissue. "The infiltrations for the most part lie quite flat, so that there is no great hindrance even though the mucosa thickens. . . . There is also no case known in which such an infiltration has led to a surgical procedure in order to make the air supply better." Suchannek,[2] in 1890, reported a case of lymphatic leukemia in which leukemic tissue infiltrated the nasal mucosa both diffusely and in circumscribed lymphomatous nodules. There was no obstruction. Sternberg[3] described, in 1921, a case of lymphatic leukemia with the most marked symptoms and signs in the tonsils; however, the nasal, laryngeal, and tracheal mucosae were also affected, presenting everywhere a pale appearance and showing some areas to be thickened by a typical infiltrative process. Menzel,[4] in 1924 reviewed one case that he had reported in 1904 and presented a new case. Both of his patients suffered from lymphatic leukemia, and both developed extensive involvement of the paranasal sinuses as well as moderate infiltration of the nasal mucosa. The nasal lesions were diffuse and smooth with intact epithelium; the mucosa and submucosa were replaced by a leukemic stroma which was most intense in the superficial layers, but extended down to the basic supportive cartilaginous or muscle tissues. Both Menzel and Sternberg urged more earnest postmortem search for upper respiratory lesions. Menzel remarked that both of his cases were of the lymphogenous form of leukemia, wondering if further observations would discover nasal and paranasal lesions of other leukemic types. In 1936, Love[5] reviewed a series of forty-one cases of leukemia with oral or otorhinoloryngeal manifestations. He stated that two patients presented leukemic infiltration of the skin and mucous membrane of the nose, but he described only one. The patient had a lymphogenous leukemia with profuse nasal discharge and complete obstruction of both nasal passages for several weeks. A biopsy of the mucosa overlying a lower turbinate showed an area of necrosis extending into the submucosa where in some areas there was a marked infiltration of immature mononuclear cells and eosinophils. It is not clear if the nasal obstruction were dependent solely upon mucosal thickening by leukemic infiltration or more upon the swelling of edema and

TABLE 1.—*Summary of Blood Studies*

| Dates | (Millions) Red Cells | (Sahli) Hgb | White Cells | Monocytes % Immature | % Pmm | Other |
|---|---|---|---|---|---|---|
| 9-18-40 | 3.23 | 56 | 28,800 | 96.7 | 0.3 | 4% lymphocytes |
| 9-23-40 | 3.50 | 71 | 13,750 | 87 | 0 | 13% lymphocytes |
| 9-25-40 | | 68 | 18,700 | 90.5 | 0.5 | |
| 9-28-40 | | | 30,150 | 88 | 0 | 12% lymphocytes |
| 9-30-40 | | 68 | 25,600 | 90 | 0.5 | |
| 10- 2-40 | | | 58,500 | 93 | 0 | 7% lymphocytes |
| 10- 4-40 | | 68 | 64,150 | 94 | 0 | |
| 10- 5-40 | | 68 | 70,950 | 96 | 0 | 4% lymphocytes |
| 10- 7-40 | | 68 | 99,300 | 96 | 0 | |
| 10- 8-40 | | 68 | 101,850 | 95 | 0 | |
| 10- 9-40 | | 68 | 100,700 | 96 | 0 | |
| 10-10-40 | | 65 | 100,300 | 96 | 0 | (Transfusion) |
| 10-11-40 | | 80 | 98,300 | 95 | 0 | |
| 10-12-40 | | | | | | (x-ray bath) |
| 10-14-40 | | 74 | 83,850 | 96 | 0 | |
| 10-15-40 | | 74 | 54,300 | 95 | 0 | |
| 10-16-40 | | 74 | 51,350 | 96 | 0 | |
| 10-17-40 | | 74 | 67,150 | 95.8 | 0.2 | 4% lymphocytes |
| 10-18-40 | | 74 | 58,100 | 95.8 | 0.2 | 4% lymphocytes |
| 10-19-40 | | 74 | 62,900 | 96 | 0.2 | (x-ray bath) |
| 10-21-40 | | 74 | 26,550 | 96 | 0 | |
| 10-22-40 | | 74 | 26,300 | 86 | 0.5 | 13.5 % lymphocytes |
| 10-23-40 | | 71 | 31,925 | | | |
| 10-24-40 | | 71 | 26,900 | 90 | 0.5 | |
| 10-25-40 | | 68 | 32,950 | 89 | 0 | 11% lymphocytes |
| 10-26-40 | | 68 | 35,200 | 91 | 0.5 | |
| 10-28-40 | | 68 | 34,900 | 91 | 0 | (x-ray bath) |
| 10-29-40 | | 65 | 24,550 | 93 | 1.0 | |
| 10-30-40 | | 62 | 31,700 | 93 | 0.5 | |
| 10-31-40 | | 57 | 43,250 | 94 | 1.0 | |
| 11- 1-40 | | 52 | 17,100 | 88 | 4.0 | (Transfusion) |
| 11- 4-40 | | 56 | 18,500 | 91 | 4.0 | |
| 11- 5-40 | | 62 | 13,450 | 94 | 3.0 | |
| 11- 6-40 | | | 13,900 | 93 | 1.0 | |
| 11- 7-40 | | 59 | 11,500 | 93 | 0 | |
| 11- 8-40 | 3.13 | 59 | 9,500 | 92 | 1.0 | 7% lymphocytes |
| 11- 9-40 | | 62 | 10,200 | 92.5 | 0.5 | |
| 11-11-40 | | | 14,050 | 89.5 | 0.5 | |
| 11-12-40 | | 65 | 12,000 | 91.5 | 0.5 | platelets 28,000 (Transfusion) |
| 11-13-40 | 3.63 | 65 | 15,600 | 93.5 | 0.5 | |
| 11-14-40 | 3.38 | 62 | 12,450 | 90 | 0 | 1% myelocytes |
| 11-16-40 | | 77 | 20,700 | 94.5 | 0.5 | |
| 11-18-40 | 3.42 | 68 | 21,000 | 88 | 0 | 1% myelocytes |
| 11-22-40 | 3.78 | 74 | 40,000 | 92.5 | 0.5 | |
| 11-25-40 | 2.86 | 56 | 17,600 | 77 | 20.0 | 3% lymphocytes |
| 11-26-40 | 2.90 | 56 | 17,400 | 60 | 28.0 | 7% lymphocytes<br>2% metamyelocytes<br>3% myelocytes<br>12% banded pmn's |

congestion related to a superimposed secondary infection. In his excellent monograph, Forkner[6] refers to the leukemic changes of the upper respiratory tract and mouth as being of two groups: the nonspecific leukemids and the specific infiltrations. Regarding the latter, he emphasizes the intensity of the infiltration particularly about the blood vessels, and states that many such lesions have been reported as occurring in the larynx and trachea. Fewer reports concern the nose and sinuses, though Forkner recalls that Viedebeck, Jaffé, and others have pointed out that localized leukemic lesions in the upper respiratory tract may constitute the first and most striking manifestations of the disease. This is well illustrated in the report of Haffly and Schipfer,[7] whose patient's presenting complaints were "pronounced bilateral nasal obstruction, progressively becoming more severe during the preceding twelve months, and a distressing sore throat of three weeks' duration." On examination it was found that "both nares were blocked by a large, bluish-gray, irregularly shaped tumor" which bled easily; by x-ray the right ethmoid cells were seen to be completely clouded and obscured by a tumefaction. Two x-ray treatments given locally over the nasal tumor resulted in its complete disappearance. Hematologic studies indicated a predominance of monoblasts, though it is stated that at autopsy all of the organs were infiltrated by a lymphoblastic type of cell. A cervical lymph node taken at biopsy was infiltrated with "a uniform, darkly staining mononuclear cell containing many mitotic figures." Classification of the case seems uncertain.

## REPORT OF CASE

*History.*—Mrs. E. M., a 61-year-old white, American woman, was admitted to the Palo Alto Hospital on September 17, 1940, a patient of Dr. Ralph D. Howe, with whom I saw her in consultation. Her chief complaint was chest pain. The present illness had started five weeks before entry, when she had suffered with a severe sore throat that persisted seven to ten days. After two weeks of freedom from symptoms the sore throat recurred; it was associated with pain in the left side of the chest posteriorly, and with a dull ache in the left upper quadrant of the abdomen. The past medical and family history were non-contributory.

*Physical Examination.*—Oral temperature, 102. The patient was in moderate distress and mildly cachectic. Her eyes, ears, and nose showed no abnormalities. The gums were of normal appearance. There was moderate redness of the posterior pharyngeal wall. No lymph nodes were palpable in the cervical region or elsewhere. Respirations were shallow and moderately rapid, and there was some splinting of the left side of the chest. Below the fifth rib, posteriorly, on the left side, there was percussion dullness; crepitant râles and a pleural friction rub could be heard in this area. The heart was not enlarged, rhythm was regular, and there was no murmur. There was guarding and tenderness in the left upper quadrant, but the spleen was not palpable. The rest of the physical examination was without positive finding.

*Laboratory.*—The urine examination was negative except for a trace of albumin. Complete blood count on September 18, 1940 showed: hemoglobin, 56 per cent (Sahli); red blood cells, 3,230,000; white blood cells, 28,800, of which 96.7 per cent were large, immature monocytic cells, 3 per cent were lymphocytes, and 0.3 per cent were polymorphonuclears.

*Diagnosis.*—Leukemia, acute, type uncertain.

*Laboratory Course.*—Between September 18, 1940, and November 22, 1940, forty-seven blood counts were made (see Table 1). The total white count ranged from 9,500 to 101,850. The differential counts revealed a predominance of large, immature, monocytic cells that had a furrowed or folded nucleus containing moderately fine chromatin structure and nucleoli; the cytoplasm was pale, finely granular, and in most instances contained fine, azurophilic granules. The hematologist, Dr. H. A. Wyckoff of the Stanford University School of Medicine, regarded these cells as monoblasts. At every examination during this period these immature cells composed from 88 to 96 per cent of the total white cell count. On the last two days of the patient's illness the total white cell count was between 17,000 and 18,000, and the differential counts revealed a decrease in the percentage of immature monocytic cells to sixty with an increase in metamyelocytes to 2 per cent, of myelocytes to 3 per cent, of banded polymorphonuclears to 12 per cent, and mature polymorphonuclears to 16 per cent. Previously the cells of the polymorphonuclear series had never exceeded 4 per cent, and usually composed less than one per cent of the total white cells. Blood platelets were counted twice and found to be between 28,000 and 30,000. The hemoglobin varied from 46 to 77 per cent (Sahli).

*Therapy.*—Four transfusions of citrated blood, 500 cubic centimeters each, were given during the three months of illness. These had no noticeable effect. A "general x-ray body bath" was given upon three occasions. Following each of these there was a marked decrease in the total number of white cells, but no alteration of the differential count.

*Clinical Course.*—The illness was one of steadily increasing weakness and decline ending with high fever, signs of generalized bronchopneumonia and death on November 26, 1940. On October 13, 1940, the spleen was easily palpated just below the left coastal margin. On October 21, 1940, the patient first noted a slight degree of obstruction in the left nostril. At this time two small lymph nodes became palpable in the posterior cervical chain, and there were other small nodes felt in the axillary and inguinal regions. Because the nasal symptoms slowly progressed to those of complete obstruction, Dr. R. Wesley Wright was called in for rhinologic consultation. The note of his examination made on November 11, 1940, is as follows: "On the left side of her nose there are two pale, irregular growths or infiltrations—one from the septum, starting almost immediately back of the vestibule; and the other from the inferior turbinate. This closes the space entirely, so that there is almost no line of cleavage between these two structures to be found there. Nothing can be inserted back farther, and there is absolutely no breathing space. . . . The right side shows a very slight beginning of pale corrugations on the septum and also on the inferior turbinate, but this side is still patent." The throat showed some slight submucosal hemorrhages around the anterior pillar on the left side where there were several small ulcerated areas. In the upper part of the pharynx there was also a small patch of early infiltration. X-ray therapy was given locally to the nasal infiltration (two treatments, five days apart), but was without effect on the appearance of the lesions or on the obstructive symptoms. The patient's general condition and her bleeding time, which was increased to ten minutes, seemed definite contraindications to any surgical procedure. On October 30, 1940, the patient developed a severe attack of herpes zoster with lesions in the distribution of the cervical nerves two and three on the left side. Several of the lesions became necrotic, and one created an ulcer about 3 by 4 centimeters in diameter on the back of the neck. Her death on November 26, 1940, came three and one-half months after the onset of her first symptoms.

*Autopsy.*—Postmortem studies were performed by Dr. Burt L. Davis. Permission for extensive examination of the head was not granted, but specimens of the nasal infiltration were obtained. The left lung at the base showed broad areas of brawny infiltration or tumor approximately two centimeters thick and covering most of the left lower lobe. The spleen was twice its normal size. Pyer's patches were prominent in the small intestine. Vertebral marrow was dark red. Other organs showed no gross abnormalities.

*Histologic Examination:*

Turbinate.—"Some of the sections show fragments of normal epithelium, but most sections show the mucosa infiltrated with irregular cells which have nuclei of various sizes and shapes, and pale staining cytoplasm. This extends downward between the mucous glands as deeply as the cartilage. Throughout the mass there are large numbers of polymorphonuclear leukocytes."

Lung.—"A section from the left base shows marked destruction of the usual pulmonary tissue. . . . Scattered through this material are numbers of large mononuclear cells which have fairly abundant basophilic cytoplasm and round to oval and, in many instances, kidney-shaped, pale-staining trabeculated nuclei; many show nucleoli, and numerous chromatin bars are seen." All other organs showed more or less infiltration with these mononuclear cells, and they were the predominant cell of the bone marrow.

## COMMENT

This case is of interest for several reasons. The review of the literature shows clearly that nasal obstruction on the basis of leukemic infiltration of the mucosa is rarely encountered. Only the case reported by Haffly and Schipfer appears directly comparable with ours, and it is perhaps worth noting that both cases were granted hematologic diagnoses of monocytic leukemia. Despite the failure of local x-ray therapy to alter the nasal lesions of our patient, it would seem still to be the treatment of choice: surgical intervention is contraindicated because of the bleeding tendency of leukemic patients, and the experience of Haffly and Schipfer shows that roentgen rays may produce excellent results. A point of minor interest is the occurrence of herpes zoster in the course of the illness. This frequent concomitant of leukemia is said to be caused by infiltration into or about the dorsal roots of the spinal cord. Two features of the case relative to the question of its classification should be remarked: (1) The unusually high and fairly constant percentage of early monocytic forms which was maintained until the last two days of life, regardless of the fluctuation

of the total white blood count; and (2) the definite change in the differential white-cell count in those final days to one showing a very appreciable percentage of immature and mature cells of the polymorphonuclear series. Those who do not acknowledge monocytic leukemia to be a distinct entity (Naegli, Piney, Kracke, Garver, and others) would perhaps point to this shift as evidence that the present case truly is one of the myelogenous type; Schilling, Sabin, Clough, and others consider these alterations in the differential count as not incompatible with a diagnosis of monocytic leukemia. Wyckoff[8] has succinctly described two types of blood picture occurring in this disease, with the conclusion that monocytic leukemia can be certainly identified. A final point of interest is that there were no troublesome oral lesions during this patient's illness, a want that is contrary to the usual experience as described by Clough and Forkner.

SUMMARY

A case of monocytic leukemia has been reported in which nasal obstruction due to mucosal infiltrations is described. A review of the literature reveals this to be a rare occurrence. Other features of especial interest in this case are discussed.

Laguna Honda Home.

REFERENCES

1. Seelenfreund, Bruno: Veränderungen der oberen Luftwege bei Leukämie, Inaug. Diss., 1925, Breslau.

2. Suchannek, Hermann: A Case of Leukemia with Noteworthy Changes of the Nasal Mucous Membrane, Arch. Otol., 19:255-269, 1890.

3. Sternberg, Hermann: Ein Beitrag zur Leukämie der oberen Luftwege, Monatsschrift für Ohrenheilkunde, 55:1655-1661, 1921.

4. Menzel, Karl M.: Veränderungen der Schleimhaut der Nasen-Nebenhöhlen bei Leukämie, Ztschr. f. Hals-Nasen- u. Ohrenh., 4:26-33, 1924.

5. Love, Andrew A.: Manifestations of Leukemia Encountered in Otolaryngologic and Stomatologic Practice, Arch. Otolaryng., 23:173-221, 1936.

6. Forkner, Claude E.: "Leukemia and Allied Disorders." The Macmillan Company, New York, 1938.

7. Haffly, Gilbert N., Schipfer, Lloyd A.: Subacute Monocytic Leukemia with Nasal Manifestations, Arch. Otolaryng., 31:858-862, 1940.

8. Wyckoff, H. A.: Monocytic Leukemia: Some Blood and Bone-Marrow Studies, Calif. and West. Med., 48: 348-349, 1938.

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Those in the general health field are apprehensive wherever social and economic factors have dislocated and reconcentrated large groups of the population. Such upheavals create what might be called an epidemic potential. One soon knows what is happening in acute communicable diseases for there is a sharp dividing line between health and acute disease. This is not the case in tuberculosis. Here the period of incubation as used in its ordinary sense, is vague, ill-defined, and long-drawn out. The onset is not dramatic and neither morbidity nor mortality figures of today reflect what is happening currently. Insofar as this disease is concerned, the aftermath of the present social and economic dislocations is as important, if not more important than the immediate effect. One must meet current problems as they arise but one must recognize that danger may not manifest itself for years to come.—Harry Mustard, M. D., *Transactions, N. T. A.*, 1943.

**George Dock Receives Distinguished Service Award**

The Distinguished Service Medal and Award of the American Association for year 1944 were conferred on Dr. George Dock, of Pasadena, Calif., famous physician and medical educator. Dr. Dock was born at Hopewell, Pa., April 1, 1860. He received the M.D. degree from the University of Pennsylvania in 1884, an honorary A.M. from Harvard in 1895, the ScD. from the University of Pennsylvania in 1904 and the LL.D. from the University of Southern California in 1936. From 1887 to 1888 Dr. Dock was assistant clinical professor of pathology at the University of Pennsylvania, becoming professor of pathology at the Texas Medical College and Hospital in 1888. He left Texas in 1891 and became professor of the theory and practice of medicine and clinical medicine at the University of Michigan, where he remained until 1908. From the latter year until 1910 he held the same title at Tulane University Medical School. He was professor of medicine at Washington University School of Medicine, 1910 to 1922, and dean part of that time. He is honorary professor of medicine at the University of Southern California and a member of numerous medical societies, including the American Medical Association, American Association for the Advancement of Science and Association of American Physicians, of which he was president in 1916-17. He is co-author of a book on hookworm disease and of articles and chapters in many textbooks on medicine. A happy conjunction of events led to the celebration of Dr. Dock's eightieth birthday at a dinner at the Los Angeles County Medical Society in April, 1940, on which occasion the organization of the Walter Jarvis Barlow Society of the History of Medicine simultaneously held its first public meeting and the birth of the George Dock Lectureship in the History of Medicine also took place, Dr. Dock being the first lecturer. The award to Dr. Dock of this honor adds a new name to the distinguished list of those who have already been recognized in this manner, including Dr. Rudolph Matas in 1938, Dr. James B. Herrick in 1939, Dr. Chevalier Jackson in 1940, Dr. James Ewing in 1941, Dr. Ludvig Hektoen in 1942 and Dr. Elliott P. Joslin in 1943.—*J.A.M.A.*, June 24, 1944.